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Spatial working memory and inhibition of return

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Recently we showed that maintaining a location in spatial working memory affects saccadic eye movement trajectories, in that the eyes deviate away from the remembered location (Theeuwes, Olivers, & Chizk, 2005). Such saccade deviations are assumed to be the result of inhibitory processes within the oculomotor system. The present study investigated whether this inhibition is related to the phenomenon of inhibition of return (IOR), the relatively slow selection of previously attended locations as compared with new locations. The results show that the size of IOR to a location was not affected by whether or not the location was kept in working memory, but the size of the saccade trajectory deviation was affected. We conclude that inhibiting working memory–related eye movement activity is not the same as inhibiting a previously attended location in space.

Working memory is a system that allows for the temporary storage of information until a task is completed (see, e.g., Baddeley, 1986). Awh and colleagues (Awh & Jonides, 2001; Awh, Jonides, & Reuter-Lorenz, 1998) provided evidence for a strong link between working memory and attention. For example, they showed that when a location is remembered, processing of stimuli at that location is facilitated relative to those at other locations (Awh & Jonides, 2001), just as attending to a location improves the processing of information at that location (Posner, 1980). Also, when visuospatial attention is disrupted during a visuospatial working memory task, the ability to remember the location is impaired (Awh et al., 1998). Finally, brain imaging studies have confirmed the notion of a strong overlap between working memory and attention, showing that the same frontoparietal network is involved in both attention and visual working memory tasks (Awh et al., 1999; Postle, Awh, Jonides, Smith, & D'Esposito, 2004). These results have been interpreted as evidence for a close link between visuospatial working memory and visuospatial attention.

In a recent study, Theeuwes, Olivers, and Chizk (2005) reported that not only does visuospatial working memory affect attention, it also directly affects the eye movement system. They determined this by looking at saccade trajectories. Whereas previous research showed that the eyes may deviate away from *visible* stimuli (see, e.g., Godijn & Theeuwes, 2002b; Sheliga, Riggio, & Rizzolatti, 1994; Van der Stigchel & Theeuwes, 2006), Theeuwes et al. (2005) demonstrated that the eyes also curve away from *remembered* stimuli. The study provided evidence for a

direct link between working memory and the oculomotor system.

Saccade curvatures have been attributed to competitive interactions of activation within intermediate layers of the superior colliculus (SC), a midbrain oculomotor structure involved in encoding stimuli as potential saccade targets (Sparks & Hartwich-Young, 1989). The SC is a lower level structure that operates as a motor map for the generation of eye movements. Its intermediate layers have direct projections to and from the posterior parietal cortex (Paré & Wurtz, 1997), a region closely related to attentional selection. Competitive interactions within SC have been shown to operate between separate populations of neural activation and are central to models of saccade curvature effects. Deviation away from a distractor has been attributed to the inhibition of distractor-related activation. This inhibition is presumably a top-down signal to prevent misdirected saccades. As a result, the overall population of activation produces a saccade vector that deviates away from the distractor location (see, e.g., Doyle & Walker, 2001; Godijn & Theeuwes, 2002b; Van der Stigchel & Theeuwes, 2005, 2006). For example, in Godijn and Theeuwes (2002b), saccade deviations were observed when observers had to make an eye movement to a predefined target while ignoring an abrupt-onset distractor singleton (referred to as the *oculomotor capture paradigm*; see Theeuwes, Kramer, Hahn, & Irwin, 1998). This creates a situation in which there is competition between endogenous and exogenous signals. The results showed that the eyes curved away from the distractor location, suggesting that the location of the distractor was inhibited in order to prevent it from capturing the eyes. The inhibition hypothesis is consistent with results from Aizawa and Wurtz (1998), who showed similar saccade trajectory deviations after local inactivation of a region of the SC.

The memory task of Theeuwes et al. (2005) required observers to remember one location (presented laterally) and then a few moments later make an eye movement to another location (i.e., straight up or straight down). In

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order to allow for an accurate saccade to the target location, the oculomotor activation associated with the remembered location needed to be inhibited. Just like the inhibition assigned to a visible distractor location, the inhibition assigned to a location in memory then caused a saccade deviation away from the memorized location.

The purpose of the present study was to determine the nature of this inhibitory process. More specifically, we addressed whether the inhibition associated with the memorized location is related to the classic inhibition effect known as *inhibition of return* (IOR; Posner & Cohen, 1984). IOR is a visual attention phenomenon characterized by delayed responses to targets presented at recently cued or recently fixated locations (see Klein, 2000, for an overview). In a typical IOR experiment, an exogenous abrupt-onset cue is presented at one of two locations. The cue is not predictive of the location of the subsequent target. Typically, detecting a target presented at the cued location is faster than detecting a target at the uncued location. However, when the interval between the abrupt-onset cue and the target is relatively long, the opposite pattern of results is observed; that is, responses to targets presented at the cued location are slower than those to the uncued location (Posner & Cohen, 1984).

In order to address the relationship between IOR and curvature deviations, in one condition we asked observers to retain the location of a laterally presented cue while executing a saccade to another location (see Figure 1). As in Theeuwes et al. (2005), this part was designed to measure saccade trajectories. To measure IOR, on some trials observers were required to execute an additional saccade to either the cued (and memorized) location or to an equidistant uncued location on the opposite side of the space. The difference in saccade latency between cued and uncued locations represents IOR. Both the trajectory deviation and the IOR effect were compared to data from a control condition in which the cue did not need to be memorized. If both the size of the deviation and the size of IOR were to increase in the memory condition relative to the no-memory condition, this would be evidence for the involvement of a common inhibitory mechanism. In contrast, if deviation and IOR were to show different effects, the two inhibitory mechanisms could then be dissociated.

METHOD

Participants

Eight students with normal or corrected-to-normal vision took part in the experiment.

Apparatus

A Pentium II computer with a 21-in. color monitor generated the stimuli and controlled the timing of the events. Eye movements were recorded by means of an Eyelink tracker with 250-Hz temporal resolution and 0.2° spatial resolution.

Stimuli, Design, and Procedure

The basic trial sequence is shown in Figure 1. Each observer was seated 75 cm from a computer screen, with head positioned on a chinrest, and fixated a center fixation dot. After 1,000 msec, a gray dot 1.35° in diameter appeared for 500 msec in one of the cells of

a 3×3 grid (measuring $2.7^\circ \times 2.7^\circ$). The grid was centered at $x = \pm 5.4^\circ$, $y = \pm 4.8^\circ$ (from the fixation point) in one of the quadrants of the display (top left, top right, bottom left, or bottom right). In one condition, the observers had to memorize the location of the dot. In the other, the dot was presented but did not need to be memorized. After a blank interval of 1,000 msec, an arrow pointing either straight up or straight down was presented for 300 msec at the center location. Depending on the direction of the arrow, an observer immediately made a saccade up or down to a visible marker placed at the top or bottom of the display, 9.5° from the central fixation dot. Note that saccades were only made to the area where the dot had been presented (i.e., to the top or bottom half of the screen). In half of the trials (randomly varied within blocks), the observer had to then make a second saccade to either the cued location (Figure 1A) or an equivalent uncued location (Figure 1B). The location to which the second saccade had to be made was indicated by a peripheral plus-sign marker that was presented 2,000 msec after the presentation of the central arrow. Upon presentation of the marker, the observer immediately made the saccade. After this sequence of events, spatial memory was tested. A white pointer (a ring of the same dimensions as the dot) was presented at exactly the same location as the initial dot or at a location deviated slightly away from the initial location, and the observers indicated with their keyboard whether or not the pointer matched the original location. When an observer committed an error, a tone sounded. Each observer performed 24 practice trials and then 144 trials without and 144 with the memory task. The order of the blocks was counterbalanced.

RESULTS

To assess the effects of memory on eye movements, we first determined the effect on saccade trajectories. We calculated the average angular deviation of the saccade path for each sample point, relative to a straight line from the starting point to the endpoint, and collapsed across quadrants (top left, top right, bottom left, and bottom right). Figure 2 gives an illustration of the calculation of the angular deviation. Note that Van der Stigchel and Theeuwes (2005) have shown that different dependent measures for saccade deviation, such as initial direction, curvature, or overall direction, all basically show the same effect. Trials on which there were no saccades or on which the saccades were too early (< 80 msec), too late (> 600 msec), or too short ($< 3^\circ$ of visual angle) were excluded from the analyses. If the endpoint of the first saccade had an angular deviation of less than 30° of arc from the center of the target, the saccade was classified as correct and further analyzed. Furthermore, the starting position for the first saccade had to be within 1° of the fixation point, and for the second saccade it had to be within 2° of the initial target. For saccade deviation, trials were removed from the analysis when the angle of the overall direction was 2.5 times the standard deviation away from the mean angle. These prerequisites led to a total loss of 24.4% of trials. An analysis on the remaining trials showed that keeping the cued location in memory caused more deviation away from the cued location than occurred when no memorization was required [the deviation was -0.03 rad (-1.73°) for no memory vs. -0.06 rad (-3.44°) for memory; $t(7) = 2.68$, $p < .05$]. This result confirms our earlier findings (Theeuwes et al., 2005). Note that the deviation in the no-memory condition was significantly different from zero [$t(7) = 2.82$, $p <$

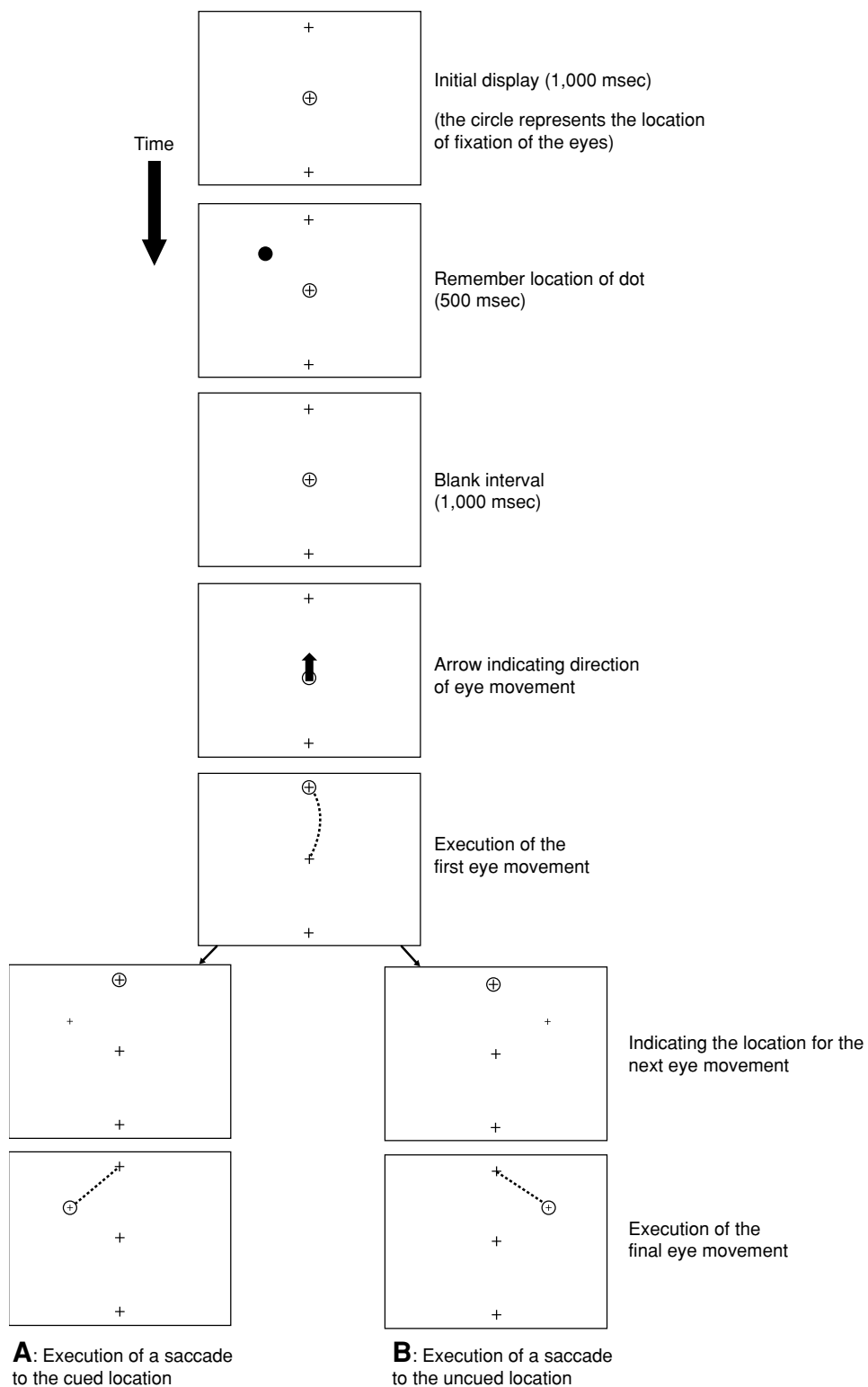


Figure 1. Typical task sequence. After the initial display, a dot was presented somewhere in one of four quadrants. In the memory condition, observers had to remember the location of the dot. In the no-memory condition, they ignored the dot. After a blank interval of 1,000 msec, an arrow indicated the direction in which a saccade had to be made (either up or down). After arriving at the indicated location, in half of the trials observers had to make a second saccade, either to the location that was held in memory (the cued location) or to an equidistant location on the other side (the uncued location).

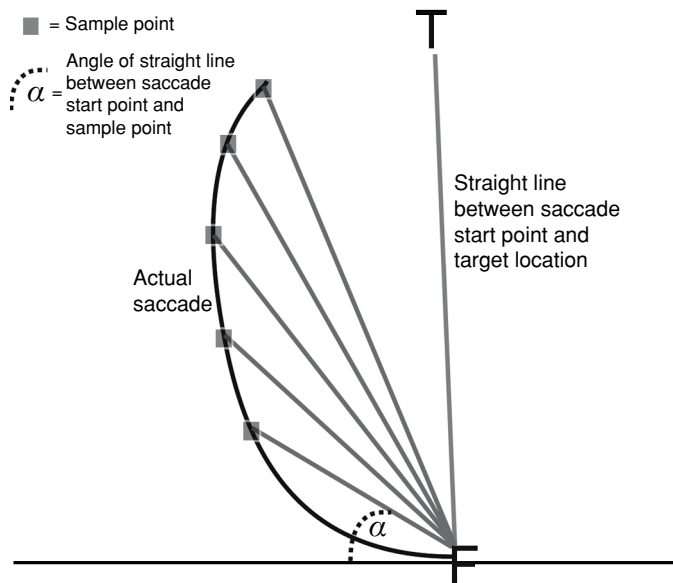


Figure 2. Illustration of the computational procedure for saccade deviation. “F” represents fixation, “T” the target. For each sample point of the actual saccade (indicated by the curved line), the angle of the straight line between the start point of the saccade and the current sample point was measured and averaged across the whole saccade. The mean angular deviation was then subtracted from the angle of the straight line from fixation to target.

.03]; nonetheless, the important point is that the deviation was significantly stronger in the memory condition than in the no-memory condition.

Second, we addressed whether memory affected IOR, as indicated by saccadic latencies of the second saccade to the cued and uncued locations. The important question was whether IOR would be modulated by whether the cued location was kept in memory or not. An ANOVA on saccade latency with the factors memory (memory vs. non-memory) and location (cued vs. uncued) showed a main effect of location [$F(1,7) = 5.75, MS_e = 720.40, p < .05$]. Figure 3 shows this effect. There was no main effect of memory, nor any interaction ($F_s < 1$). As is clear from this figure, the mere presentation of the dot resulted in an IOR effect of about 20 msec, and this effect did not depend on whether the location of the dot was kept in memory or not. The crucial point is therefore that keeping a location in memory does affect the saccade trajectory, but it does not have a modulating effect on the size of IOR.

On average, the observers made 22.3% errors in the memory task. There was a trend [$t(7) = 1.72, p = .13$] for memory performance to be better when a second saccade had to be made (19.7% errors) than when no second saccade had to be made (24.8%). However, in the condition in which no second saccade had to be made, a memory performance of 75.2% was still found, which is well beyond chance (50%). This implies that observers followed the instructions and kept the location of the dot in working

memory during the execution of the saccades until they had to perform the memory task.

Note that the latency of the initial saccade did not differ between the memory and no-memory conditions (330 vs. 331 msec; $F < 1$), indicating that it did matter whether observers had to perform the current task with or without a concurrent memory load.

DISCUSSION

The present study confirmed our earlier finding (Theeuwes et al., 2005) that keeping a location in memory causes the eyes to deviate away from that location. The important new conclusion is that the size of IOR is not affected by whether the location is kept in working memory or not. Thus, inhibiting a location in spatial working memory is not the same as inhibiting a previously attended location in real space, the phenomenon referred to as IOR.

In the present experiment, an abrupt-onset dot was presented at a peripheral location in the visual field. It is known that an abrupt onset captures attention in an exogenous way (see, e.g., Theeuwes, 1991); it has also been shown that such abrupt exogenous stimuli lead to IOR (e.g., Posner & Cohen, 1984), and the same effect was observed here. IOR occurred with equal magnitude whether or not a location was kept in memory, yet keeping that location in memory did systematically influence the saccade trajectory so that the eyes deviated away from the

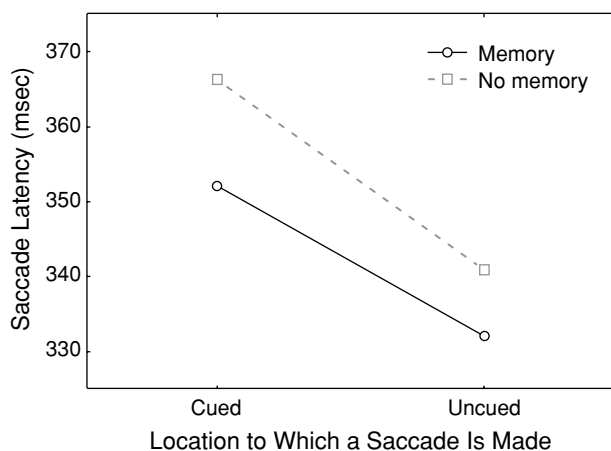


Figure 3. Latencies for the second saccade toward the cued and uncued locations, for conditions in which the cued location was or was not held in memory.

memorized location. These results suggest that the inhibitory mechanisms causing saccade deviations differ from those associated with IOR.

The present findings can be explained by considering the architecture of the oculomotor system. Godijn and Theeuwes (2002a, 2002b; Theeuwes & Godijn, 2004; for a similar model, see Trappenberg, Dorris, Munoz, & Klein, 2001) proposed a competitive-integration model in which saccades are programmed in a common saccade map (most likely the SC) in which activations from different sources (e.g., exogenous and endogenous) are integrated. Deviations in saccade trajectories are the result of competitive interactions of activation within intermediate layers of the SC involved in encoding stimuli as potential saccade targets (Sparks & Hartwich-Young, 1989)—interactions that, according to Godijn and Theeuwes (2002b), are further affected by top-down inhibition of potential distractors. When an abrupt onset is present in the display to draw attention and an eye movement has to be executed immediately, the exogenous activation caused by the abrupt onset needs to be suppressed to prevent it from capturing the eyes. The saccade direction is ultimately determined by the mean vector of these activities, and it may curve away from the distractor (Godijn & Theeuwes, 2002b; Lee, Rohrer, & Sparks, 1988; Tipper, Howard, & Paul, 2001).

Godijn and Theeuwes (2004) also showed that the exogenous activation caused by an abrupt-onset distractor is short-lived, with saccade deviations disappearing after 800 msec. Thus, exogenous activation induced by an abrupt onset causes saccade deviations, but only when observers are concurrently set to perform an eye movement. This is consistent with the present findings from the no-memory condition, in which the abrupt-onset dot was presented but did not need to be memorized. After the relatively long delay of 1.5 sec between the presentation of the abrupt-onset dot and the arrow that instructed observers to execute a saccade, there was basically no effect of the abrupt onset on saccade deviation. However, cru-

cially, keeping the location in memory did cause a saccade deviation, even after the long 1.5-sec delay, suggesting that even though the exogenous activation of the abrupt onset may have worn off, keeping the location in memory generated enough activation in the oculomotor system to induce a saccade deviation.

Furthermore, consistent with the present findings, Godijn and Theeuwes (2004) found that the time courses of saccade curvature and IOR can be dissociated. They showed that saccade trajectory deviations are optimal immediately after the abrupt onset, but IOR became optimal only after a delay of about 800 msec from the abrupt onset. Godijn and Theeuwes (2004; Theeuwes & Godijn, 2004) presented a framework to explain these different inhibitory mechanisms that seem to operate within different subsystems. This framework consists of three subsystems: a preoculomotor attentional map, a saccade map, and an inhibitory control system. Within the saccade map (most likely in the superior colliculus), the final stage of saccade programming takes place. Activation within the saccade map then generates activation in the inhibitory control system (most likely dorsolateral prefrontal cortex and/or the frontal eye fields). Activation in the inhibitory control system in turn generates an inhibitory tag within the preoculomotor attentional map (most likely the lateral intraparietal area). The present data are consistent with this architecture, which can account for the two inhibitory mechanisms. IOR is caused by automatic exogenous activation that is delivered to the saccade map. The abrupt dot in the present experiment captured attention and caused exogenous activation within the preoculomotor attentional map and the saccade map, corresponding to the location in space where the stimulus was presented. In turn, because of its exogenous nature, the onset generated an inhibitory tag delivered to the inhibitory control system corresponding to the location of activation within the saccade map. This inhibitory tag generated by the short-lived exogenous activation of the abrupt onset caused the attentional inhibition typically referred to as IOR: After attention is reflexively shifted to the location of the initially presented stimulus, there is delayed responding to stimuli subsequently displayed at that location. Note that recent evidence has confirmed the notion that IOR is not the result of inhibition applied to the saccade map (in the SC) but is caused by inhibition on processes preceding the saccade map (Dorris, Klein, Everling, & Munoz, 2002).

The saccade curvature is caused by top-down, location-specific inhibition applied directly on the activation of a specific location. As noted, the location-specific inhibition resolves the competition when two distant locations within the saccade map are activated. The initial exogenous activation within the preoculomotor attentional map caused by an abrupt-onset dot is short-lived and fades over time. Note that this short exogenous activation causes an inhibitory tag delivered to the inhibitory control system that ultimately will result in IOR. Thus, when an observer does not need to keep the location in memory, the short burst of exogenous activity in the SC will fade. By the time a saccade has to be made, there is no need to actively inhibit

this activity in the SC, and therefore the saccade trajectory is hardly affected. However, when observers have to keep the location in memory, the initial short-lived exogenous activation caused by the abrupt-onset dot is taken over by more sustained endogenous activation. Given previous findings, it is reasonable to assume that the preculo-motor attentional map (the lateral intraparietal area) is able to sustain top-down attentional activation (see, e.g., Bisley & Goldberg, 2003). Because of an instruction to memorize the location of a dot, the top-down activation within the saccade map will need to be inhibited. It is assumed that dorsolateral prefrontal cortex and the frontal eye fields are responsible for inhibiting a specific location in the SC (e.g., Chelazzi & Corbetta, 2000; Pierrot-Deseilligny, Rivaud, Gaymard, & Agid, 1991). This location-specific inhibition causes saccades to deviate away from the location that is kept in memory.

On the basis of this model, we assume that IOR is the result of the initial short-lived exogenous activation caused by the initial abrupt-onset dot, but the saccade deviations observed in the memory condition are the result of inhibiting the sustained endogenous activation necessary to keep the location in memory.

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